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IF IF THERE IS LV MYOCARDIAL FIBROSIS, SHOULD WE EXPECT TO FIND RV MYOCARDIAL FIBROSIS? A CARDIOVASCULAR MRI STUDY

ACC Poster Contributions
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Introduction: CMR has become theideal technique to detect hypertrophic cardiomyopathy (HCM) providing complete coverage of both ventricles with high spatial resolution. Late gadolinium enhancement (LGE) accurately identifies regions of myocardial fibrosis. Via CMR, innumerable studies have established LVH as the predominant phenotypic expression. It is well known that myocardial fibrosis can occur in pts with HCM and is independently linked to a poorer prognosis than those without fibrosis by CMR. The genotypic expressions would appear to affect the entire heart yet, classically, descriptions have been limited to the LV, in some part due to the inability to image the smaller, thinner RV. Thus, little information is available about the RV in HCM.

Hypothesis: We hypothesize there is significant RV involvement in HCM when incorporating CMR analysis for RV hypertrophy and fibrosis.

Methods: Review of all pts referred for HCM was performed. SSFP/LGEE was used to diagnose patients with HCM, using gadolinium administration (0.15mmol/kg, MultiHance, Bracco Diagnostics, Princeton, NJ). Post-injection LGE images were obtained via T1-weighted, IR preps. Regions of myocardium with abnormally high signals (>2SD) were designated as fibrotic. LV/RVMass Index was calculated.

Results: Via 73 pts referred for HCM from 2006-2010, 19 (26%) were CMR confirmed. Image quality was judged excellent in18/19 (95%). The mean LVMI was 94±37gm2 (> 2SD > normal) while the mean RVMI was 21±6 gm2 (1SD > normal). All pts met formal LVH criteria while 10/19 (53%) met RVH criteria. 16/19 (84%) had evidence of LV fibrosis while 13/19 (68%) had evidence for RV fibrosis. No pt with RV fibrosis had absent LV fibrosis. In neither the LV nor RV was there a difference in the degree of hypertrophy as to prediction of likelihood for fibrosis (99±40 vs.83±27g/m2 and 21±25 vs.21±26g/m2, respectively).

Conclusions: The high frequency of RV fibrosis in the setting of HCM is surprising in that this phenomenon is rarely described. However, given the genetic abnormalities, there is no reason to expect the phenotypic expression should be limited to just the LV. Interestingly, as for the LV, the degree of RVH had little predictive power to define RV fibrosis.